PURPURA IN THE NEWBORN

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In the newborn infant, petechiae with a distribution limited to the head and upper chest are not uncommon and are due to a temporary increase in venous pressure during delivery. In contrast, generalised purpura is rare and of the many possible causes, perhaps the most common is thrombocytopenia associated with idiopathic thrombocytopenic purpura (I.T.P.) in the mother. One such case was encountered recently.

The child was born from a 28-year old primigravida who in 1954, at the age of twelve years, had undergone splenectomy for I.T.P. At that time, she presented with a history of easy bruising and petechiae since the age of seven and at the menarche (at twelve years) there had been profuse menstrual bleeding which required blood transfusion. Her platelet count then had been around 4000/cu.mm. and after splenectomy it had risen to 78000/cu.mm. *(Fig. 1)*. Subsequently, her tendency to easy bruising had diminished and her menstrual flow had never been excessive.

The pregnancy was uneventful except for three episodes of slight gingival bleeding and occasional crops of generalised petechiae. She received no drugs except vitamins and minerals (Pregnavite). A blood test done when she was 34 weeks pregnant gave the following result: Hb: 13g% (88%), W.B.Cs: 15,500/cu.mm., N: 74%, L: 15%, M: 10%; Platelets: 50,000/cu.mm. Film showed normochromic R.B.Cs and no immature leucocytes. The bleeding time was 9 minutes and clotting time 5 minutes.

The child was born on 8.5.71 at the Blue Sisters Hospital by Caesarean section because of prolonged labour and anterior second degree placenta praevia. Two pints of blood had to be given because of bleeding before and during section. At operation the uterus was noted to be free from any subserous haemorrhages or petechiae and coagulation appeared normal. After delivery it was noted that the mother had petechiae over the face and the neck and scattered bruises. A repeat blood test done on the fourth post-operative day gave Hb: 9.6g% (65%), P.C.V. 33%, W.B.Cs: 48,000/cu.mm., Platelets 100,000/cu.mm. The film showed slight anisocytosis of normochromic R.B.Cc. The W.B.Cs were normal but some of the platelets were large and mostly agranular.

The male infant, weighing 3.6 kg (8 lb.), cried straightaway at birth. On examination he had generalised purpura and areas of ecchymosis *(Fig. 2)*. There was no hepatosplenomegaly. The femoral vein puncture site bled more briskly than usual and the Hess tourniquet test was positive. The report on the blood drawn at birth was: Hb: 14.5 g% (98%), W.B.Cs 23,5000/cu.mm.. N: 65%, L: 26%, M: 8%, Platelets: 20,000/cu.mm. Film stowed slight hypochromia and macrocytosis, few poikilocytes and basophilic R.B.Cs. There were no normoblasts and no immature WBCs.

The child was given 1 mg. Vitamin K_1 i.m. and nursed in an incubator. Although his general condition was satisfactory, his sucking reflex was poor and he had to be tube-fed for the first four days. On the fourth day of life the platelet count was found to be less than 10,000/cu.mm. and the bleeding time was considerably prolonged. Because of the real danger of intracranial haemorrhage the infant was started on oral Prednisone 15mg daily (2mg/lb), with gradual lowering of the dose over the next seven days. In spite of this, the platelets continued to drop to an alarmingly low count and the dosage had to be stepped up again and continued

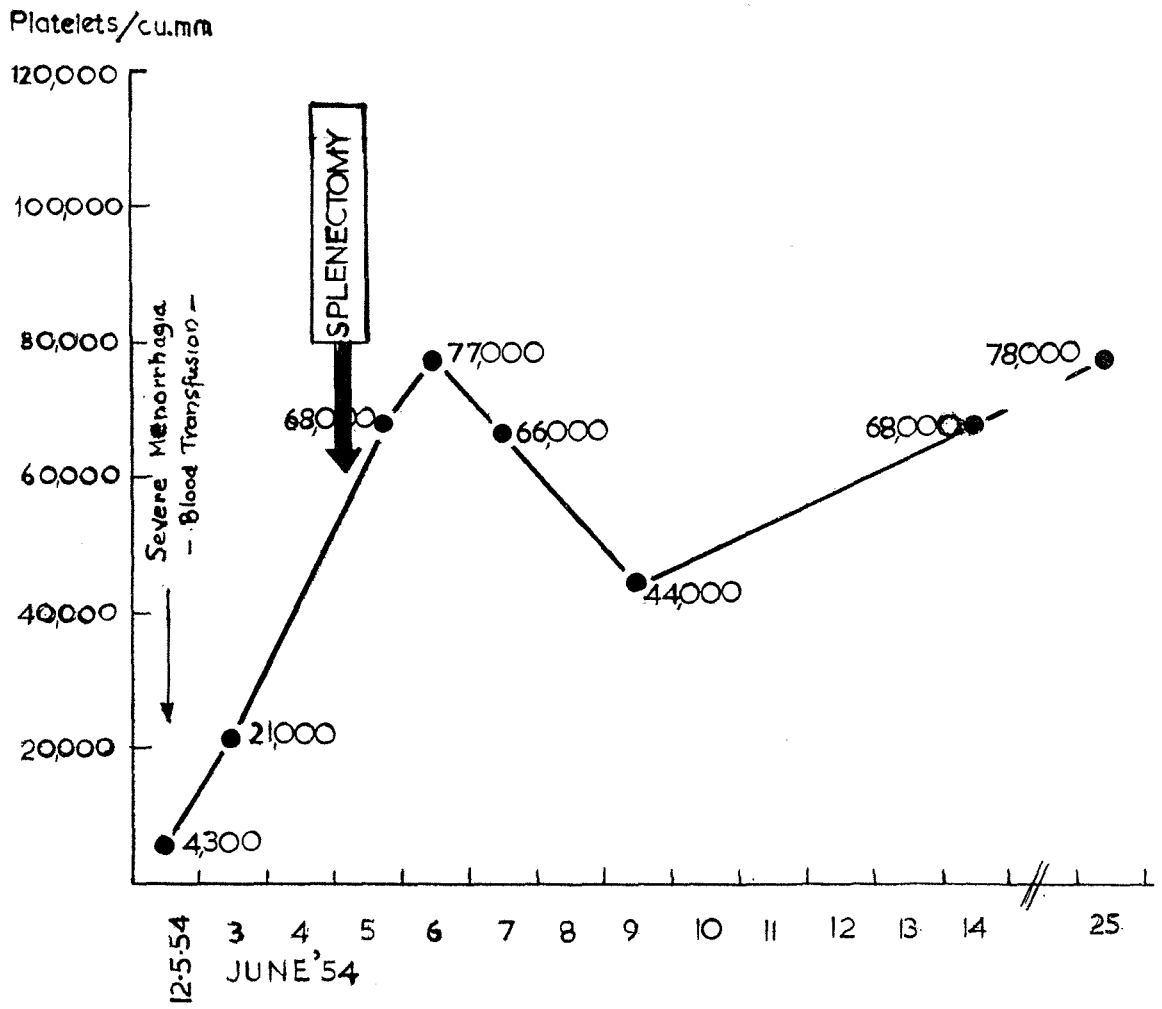

Fig.1 Idiopathic Thrombocytopenic Purpura in Infant's mother - treated by splenectomy when she was 12years old.

for over eight days *(Fig. 3)*. By the eighteenth day of life the count had gone up to 30,000/cu.mm. and the Prednisone was then gradually diminished by 5mg. every two weeks and finally stopped. The platelet count at three months of age was around 100,000/cu.mm.

Comments

The causes of neonatal thrombocytopenia are numerous *(see Table)*, but the disorders in which thrombocytopenia may occur in both mother and infant are only three, viz: idiopathic thrombocytopenic purpura (I.T.P.), drug-induced purpura and systemic lupus erythematosus.

The association of I.T.P. in the mother and her child and the demonstration of platelet agglutinins in both showed that the condition had an immunological basis (Jones *et al.*, 1961). It is due to the passage of platelet antibodies from the mother across the placenta to the child resulting in immunological destruction of the infant's platelets and neonatal purpura. The subject of I.T.P. in pregnancy has been well reviewed by Goodhue and Evans (1963) who found that the most important factor in determining whether the

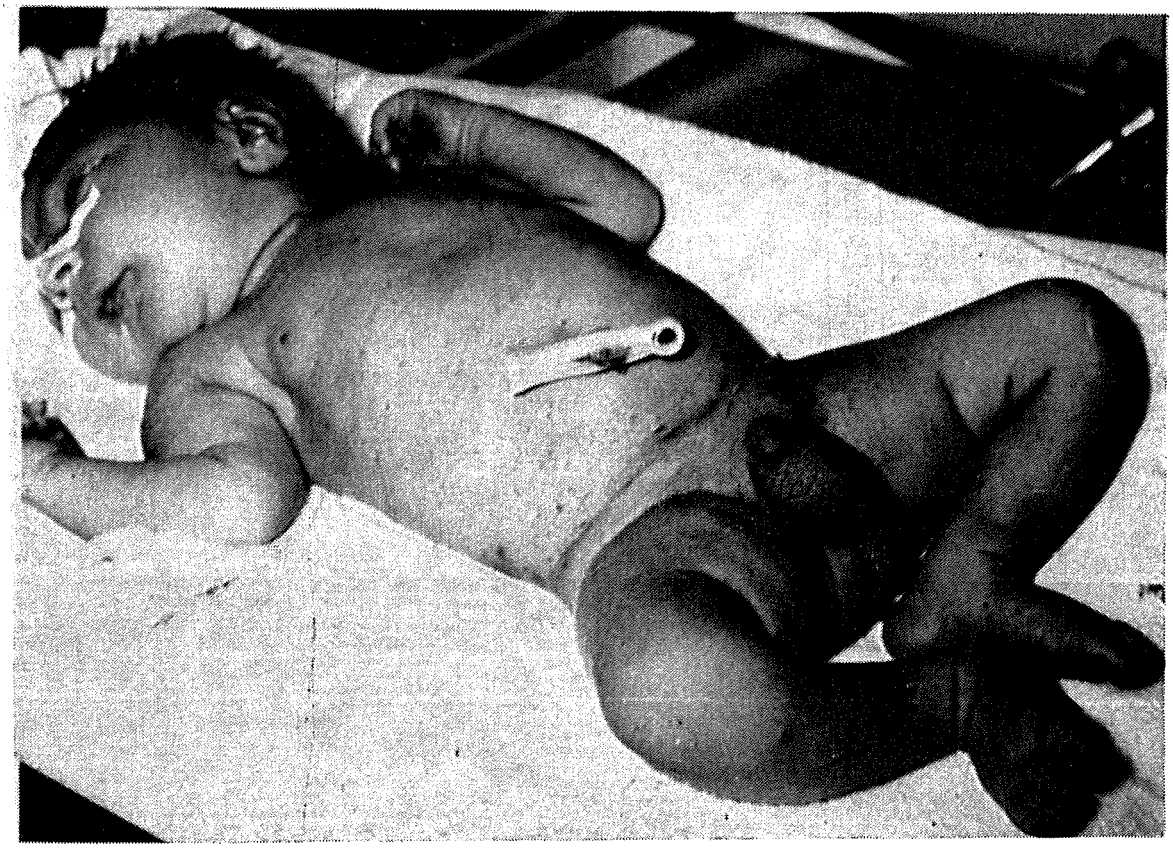

Fig. 2

infant of a mother with I.T.P. will be affected or not is the stage of activity of the mother's disease at the time of delivery. If the platelet count is normal, then the chances are that the infant will not be affected. However, it is still possible for the maternal platelet antibody to persist in mothers who have undergone splenectomy or who are in clinical remission, thus causing thrombocytopenia in their offspring.

Neonatal thrombocytopenia presents as generalised petechiae, purpuric spots and bleeding from various sites, e.g. maelena, haematuria and umbilical cord haemorrhage. Usually, the sole danger is from intracranial bleeding, which accounts for the estimated mortality of 8-10% (Anthony and Krivit, 1962). The maximal signs of bleeding occur soon after birth but in mild cases there may be minimal, delayed or no bleeding at all, in which case the only abnormally would be thrombocytopenia. The low platelet count may persist for up to four months but the risks of haemorrhage are greatest in the first week of life. Serological tests for platelet antibodies are difficult to perform and to interpret.

In most cases no treatment is needed as the condition is mild and resolves spontaneously. With severe thombocytopenia, one must treat actively because of the threat of intracranial bleeding. The methods of treatment available are: exchange transfusion with fresh blood (to remove platelet antibody from the infant), platelet transfusion and corticosteroids. Opinions differ as to the value of the latter, but whatever the treatment used it is difficult critically to evaluate its effectiveness because of the marked variations in the clinical severity of this condition. It is not surprising, therefore, that the value of steroids is still unproven. Splenectomy is a definite contraindication in young children because of the risk of overwhelming infection.

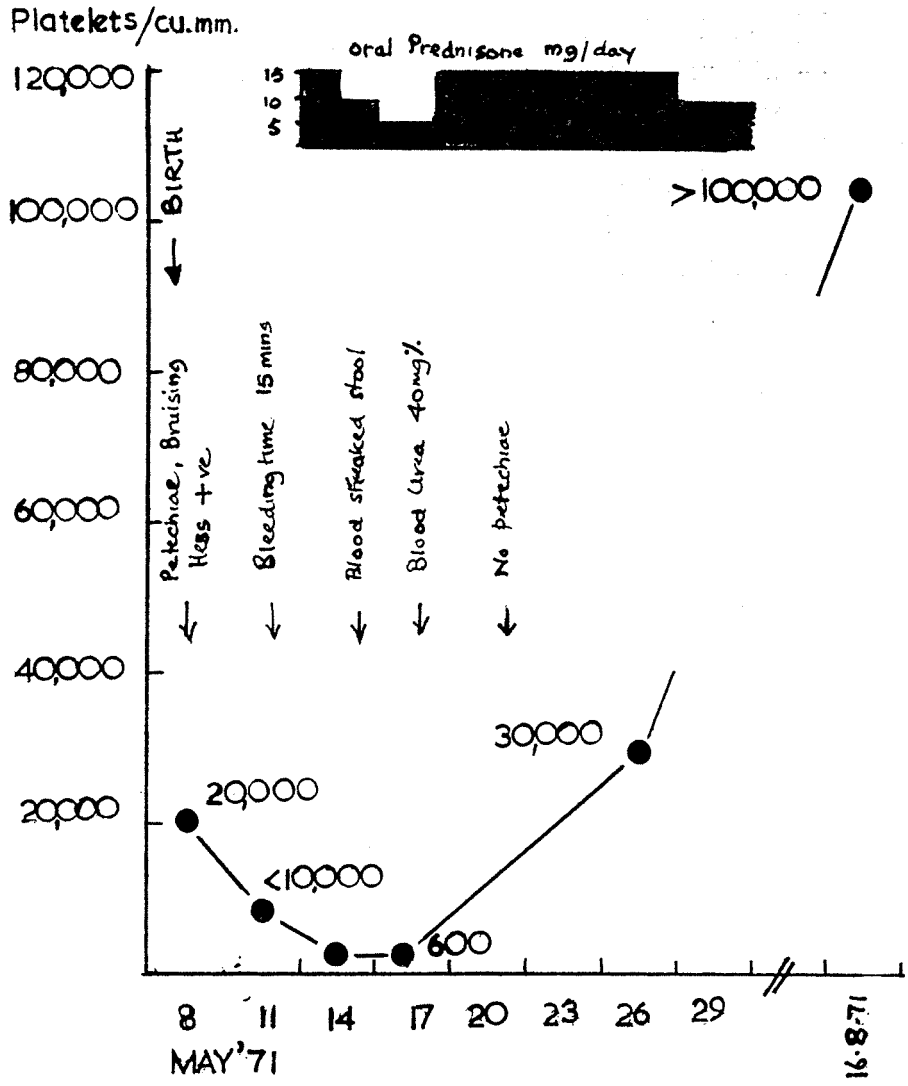

Fig.3 Thrombocytopenic Purpura in Neonate - Steroid Treatment.

Causes of Neonatal Thrombocytopenia
(modified from Oski and Naiman 1966)

(1) Immune disorders:
- (a) *Passive* — I.T.P.
  - — Drugs e.g.: Sulphas, Quinidine.
  - — Systemic lupus erythematosus.
- (b) *Active* — Isoimmune: Platelet-group incompatibility
  - — Associated with Rhesus Incompatibility.

(2) Infections:
- (a) Bacterial — Septicaemia
  - — Syphilis
- (b) Viral — Rubella
  - — Herpes simplex
  - — Cytomegalovirus
- (c) Protozoal — Toxoplasmosis

(3) Bone Marrow defects:
- (a) Megakaryocytic Hypoplasia
  - i) Isolated — Congenital hypoplastic thrombocytopenia.
  - ii) With Congenital Anomalies —

— absent radii
— rubella syndrone
— with pancytopenia (Fanconi's anaemia).

(b) Congenital Leukemia

(4) Hereditary Thrombocytopenias:

(a) Sex-Linked — including Aldrich's syndrome

(b) Autosomal — dominant and recessive forms.

(5) Miscellaneous:

i) Giant Haemangioma

ii) Disseminated Intravascular Coagulation.

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